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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
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EXAMINER	
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ART UNIT	PAPER NUMBER
1633	

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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/698,190	GRIMPE ET AL.	
	Examiner	Art Unit	
	SCOTT LONG	1633	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *19 March 2009*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1,4,7,10-17,21-28,35 and 37-54* is/are pending in the application.

4a) Of the above claim(s) *4,7,10,11,14-16,21,22,27,35 and 37-54* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1,12,13,17,23-26 and 28* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

The examiner acknowledges receipt of Applicant's Remarks and Claim amendments, filed on 19 March 2009.

Claim Status

Claims 1, 4, 7, 10-17, 21-28, 35 and 37-54 are pending. Claims 2-3, 5-6, 8-9, 18-20, 29-34, 36, 55-56 are cancelled. Claims 4, 7, 10-11, 14-16, 21-22, 27, 35, 37-54 were withdrawn by the examiner in the previous Office Action, as being drawn to non-elected inventions. Claims 1, 12-13, 17, 23-26 and 28 are under current examination.

Priority

This application claims benefit from provisional U.S. Application No. 60/423,082 filed 1 November 2002 and claims benefit from provisional U.S. Application No. 60/471,447 filed 16 May 2003. The instant application has been granted the benefit date, 1 November 2002 from the application 60/423,082.

RESPONSE TO ARGUMENTS

Claim Objections

The objection to claim 17 is withdrawn in response to the applicant's claim amendment. The amendment to claim 17 has introduced a comma between "oligonucleotides" and "ribozymes" in line 4 of the instant claim.

35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

The factual inquiries set forth in *Graham* **v.** *John Deere Co.*, 383 U.S. 1, 148 USPQ 459 (1966), that are applied for establishing a background for determining obviousness under 35 U.S.C. 103(a) are summarized as follows:

1. Determining the scope and contents of the prior art.
2. Ascertaining the differences between the prior art and the claims at issue.
3. Resolving the level of ordinary skill in the pertinent art.
4. Considering objective evidence present in the application indicating obviousness or nonobviousness.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation

under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

Claims 1, 12-13, 17, and 23-26 remain rejected under 35 U.S.C. 103(a) as being obvious over unpatentable over Fawcett et al. (Brain Research Bulletin. 1999; 49(6): 377-391) in view of Kleesiek (WO01/49831) and further in view of Jen et al. (Stem Cells 2000; 18:307-319) for the reasons of record and the comments below.

The applicant's arguments have been fully considered but are unpersuasive.

The applicant makes three specific arguments:

(a) The cited art does not teach or suggest that inhibiting expression or activity of XT-I or XT-II using nucleic acid agents (RNAi, DNAzymes, Ribozymes, antisense) can and/or will reduce glycosaminoglycan content in a glial scar of a mammal (Remarks, page 13). Contrary to the applicant's arguments, the cited art suggests such a strategy. Fawcett suggests preventing synthesis of glycosaminoglycan (GAG) in a glial scar can promote neural regeneration (page 384, *How Might Axon Regeneration Be Promoted?* and *Preventing Synthesis of Inhibitory Molecules* sections). Kleesiek teaches XT is an enzyme in the biosynthesis of the glycosaminoglycan. A skilled artisan, knowing that preventing synthesis of glycosaminoglycan in a glial scar can promote neural regeneration, would seek methods to inhibit glycosaminoglycan. A skilled artisan might ask himself how he could inhibit GAG expression. A skilled artisan in seeking such information would come upon Kleesiek. Kleesiek teach the nucleic

acid sequences of XT-I and XT-II and further suggest methods of gene therapy and methods of inhibiting XT-I and XT-II. A skilled artisan knowing that inhibiting XT-I and/or XT-II is capable of inhibiting the synthesis of GAG would seek methods of inhibiting XT-I and/or XT-II; agents used in gene therapy methods such as DNAzymes would be among those considered by a skilled artisan. Jen et al. is a review article about designing antisense oligonucleotides, ribozymes, and DNAzymes in which Jen et al. teaches "the DNAzyme can be made to cleave virtually any RNA that contains a purine-pyrimidine junction" (page 312, col.2). A skilled artisan having the knowledge of Jen and Kleesiek and Fawcett would be able to make a DNAzyme to inhibit XT-I and/or XT-II, with the knowledge that these molecules would be agents that could inhibit GAG formation in glial scars. Accordingly, the examiner concludes that a skilled artisan would find that the cited art suggests inhibiting expression or activity of XT-I or XT-II using nucleic acid agents (RNAi, DNAzymes, Ribozymes, antisense) can and/or will reduce glycosaminoglycan content in a glial scar of a mammal. Accordingly, the examiner finds this particular argument unpersuasive.

(b) The Office Action provides no teaching or suggestion of intrathecal, topical or local administration of agents to the glial scar (Remarks, page 15). The Jen article discusses the issues required for RNAi, DNAzymes, Ribozymes, and antisense drugs to provide therapeutic effects, including drug delivery and localization. A skilled artisan, knowing the difficulties in delivering drugs to neurons and the requirement that the nucleic acid drugs listed above would require co-localization within the same intracellular compartments as the mRNA being targeted, would choose routes of

administration such as intrathecal, topical or local administration. Therefore, the examiner concludes that a scientifically logical rationale exists for intrathecal, topical or local administration of RNAi, DNAzymes, Ribozymes, and antisense drugs. Therefore, the examiner finds the applicant's arguments unpersuasive.

(c) Fawcett et al. teach away from inhibiting expression or activity of XT-I and XT-II as a means to reduce glycosaminoglycan content in a glial scar of a mammal. In support of the applicant's view, he quotes Fawcett who describes some of the drawbacks (i.e., toxicity) of using certain proteoglycan synthesis inhibitors. Despite these comments by Fawcett discussing alternative strategies of inhibiting proteoglycan synthesis, Fawcett makes clear that preventing synthesis of proteoglycans is a potential method of reducing glial scars and promoting axonal regeneration. Fawcett offers only a couple of ideas for preventing synthesis of proteoglycans which include (1) blocking cytokines produced by glial scars and (2) treatment with chlorate and xylosides. While Fawcett et al. point a skilled artisan in the direction of preventing synthesis of proteoglycans as a method for reducing glial scars and promoting neuronal regeneration, Fawcett does not suggest the method of inhibiting glial scarring caused by proteoglycan synthesis with administration of RNAi, DNAzymes, Ribozymes, and antisense drugs. However, there is nothing in Fawcett that indicates that inhibiting glial scarring caused by proteoglycan synthesis with administration of some agent would not be successful. Rather, the teachings of Fawcett would encourage a skilled artisan to look for alternative (i.e., non-toxic) approaches to inhibiting glial scarring caused by

proteoglycan synthesis. Therefore, the examiner finds the applicant's argument unpersuasive.

The applicant has suggested that the examiner should consider the objective evidence of non-obviousness (page 17). The applicant directs the examiner's attention to portions of the specification and post-filing art by the instant inventors, namely the Brain (2008) abstract, which demonstrate successful practice of the *in vivo* methods of claims 1 and 17. The examiner acknowledges that at the time of filing none of the references cited in the pending rejection demonstrated the success achieved by the inventors in the Brain (2008) article and in Example 8 of the instant application. In fact, the examiner has never asserted that the cited art has demonstrated the success of the claimed method. Rather, the examiner has asserted that a skilled artisan would have taken guidance from the cited art to devise a method as claimed. Throughout the prosecution of the instant application the examiner has considered several other successful methods of reducing glycosaminoglycan content in a glial scar by administering an agent. These encompassed "inhibiting expression of primary proteoglycans" and "inhibiting chain elongation enzymes." So the examiner concludes that a skilled artisan would be successful in practicing a methods of reducing glycosaminoglycan content in a glial scar by administering an agent that inhibits expression of a chain initiation enzyme where the agent is one of RNAi, DNAzymes, Ribozymes, and antisense drugs. There seems to be nothing in the art or presented during the prosecution which indicates that there would not be success in practicing the invention suggested by the cited art and practiced in Example 8 and Brain (2008).

Furthermore, actual demonstration of success does not exhibit an unexpected result. If two other methods of inhibiting GAG were successful, then the method as currently claimed should also be successful. Therefore, the examiner finds the applicant's argument unpersuasive.

The applicant further argues claims 26-27 are allowable because they were not properly addressed in the Office Action. Claims 26-27 are directed to the method of claim 25 and further specify neurorophic factors and growth factors. In the Restriction requirement filed 6/7/2006, the examiner required a species election for the growth factors or neurotrophic factors. In the applicant's response (filed 9/6/2006) to the Species Election requirement, the applicant elected "nerve growth factor." As claim 27 is directed to bFGF, it should properly be withdrawn. While Fawcett indicates that several different growth factors can be used to help regenerate neurons, none of the cited art specifically recite using nerve growth factor (NGF). However, as the scope of the method is involved in regenerating neurons, the examiner asserts a skilled artisan would use neurotrophic factors, such as nerve growth factor in a method of inhibiting glial scars and promoting neuronal regeneration because NGF induces the differentiation and survival of neurons and is critical for the survival and maintenance of sympathetic and sensory neurons. Therefore, the examiner finds the applicant's argument that claims 26-27 are allowable to be unpersuasive.

Therefore, the examiner hereby maintains the rejection of claims 1, 12-13, 17, and 23-26 remain rejected under 35 U.S.C. 103(a) as being unpatentable over Fawcett et al. in view of Kleesiek and further in view of Jen et al.

The examiner reiterates the pending rejection:

Claims 1, 12-13, 17, and 23-26 are rejected under 35 U.S.C. 103(a) as being unpatentable over Fawcett et al. (Brain Research Bulletin. 1999; 49(6): 377-391) in view of Kleesiek (WO01/49831) and further in view of Jen et al. (Stem Cells 2000; 18:307-319).

Claim 1 is directed to a method of reducing glycosaminoglycan (GAG) content in a glial scar of a mammal comprising administering to the glial scar of the mammal an agent that inhibits the expression and/or activity of a chain initiation enzyme wherein the agent is selected from the group consisting of antisense oligonucleotides, ribozymes, DNA enzymes, and RNAi constructs, the agent targeting a nucleic acid sequence encoding xylotransferase I (XT-I) or xylotransferase II (XT-II); wherein the agent is administered intrathecally, topically, or locally to the glial scar.

Claim 17 is directed to a method of promoting neuronal regeneration in a subject comprising administering an agent to the to a nervous system lesion to inhibit a GAG chain initiation enzyme, wherein the agent is selected from the group consisting of antisense oligonucleotides, ribozymes, DNA enzymes, and RNAi constructs, the agent targeting a nucleic acid sequence encoding xylotransferase I (XT-I) or xylotransferase II (XT-II); wherein the agent is administered intrathecally, topically, or locally to the nervous system lesion; wherein the neuronal regeneration includes neurite extension into the nervous system lesion.

The remaining claims are directed to the agent being a DNA enzyme (claims 12 and 23) and wherein there is an additional administration of a growth factor or

neurotrophic factor (claim 25). Claims 13 and 24 are directed to specific DNA enzymes SEQ ID NO:33 and 39.

Fawcett et al. teach damage to the CNS results in formation of glial scars (abstract) and chondroitin sufphate glycosaminoglycan (GAG) expression is increased around the glial scars of CNS injury (page 382, col.1, lines 1-10) and that GAG expression around glial scars inhibit axon growth (page 382, col.2, lines 8-15). Fawcett et al. teach “disruption of proteoglycan synthesis…has been shown to reduce inhibition [of glial growth]” (page 382, col.2, lines 10-11). Fawcett et al. teach “How might axon regeneration be promoted?...If one wishes to reduce the influence of inhibitory molecules how might one do it? Essentially the options are to remove the cells that produce them, to reduce their synthesis, to block their activity, or to degrade them." (page 284, col.1).

Fawcett et al. do not specifically suggest using antisense oligonucleotides, ribozymes, DNA enzymes, or RNAi constructs to inhibit XT-I or XT-II.

Kleesiek teaches cloning of cDNA of human and rat xylotransferase-I and xylotransferase-II (XT-I and XT-II) and expression of recombinant proteins (abstract). Kleesiek teaches XT is the initial step enzyme in the biosynthesis of the glycosaminoglycan linkage region. (page 2, lines 8-9). Kleesiek teaches “knowledge of the cDNA sequence of XT allows to use it on gene level such as in gene diagnostic or gene therapy" (page 2, lines 25-26). Kleesiek suggests making medicaments which are inhibitors of xylosyltransferase (page 17, lines 3-4).

Kleesiek does not specifically teach using antisense oligonucleotides, ribozymes, DNA enzymes, or RNAi constructs to inhibit XT-I or XT-II.

Jen et al. is a review article about designing antisense oligonucleotides, ribozymes, and DNAzymes. Jen et al. teaches "the DNAzyme can be made to cleave virtually any RNA that contains a purine-pyrimidine junction" (page 312, col.2). The examiner believes that this teachings along with the teachings of Kleesiek which describe the DNA sequence for xylotransferase-I and xylotransferase-II, make any DNA enzyme obvious.

Claim 26 is directed to the method of claim 25 and further requiring administration of nerve growth factor. While Fawcett indicates that several different growth factors can be used to help regenerate neurons, none of the cited art specifically recite using nerve growth factor (NGF). However, as the scope of the method is involved in regenerating neurons, the examiner asserts a skilled artisan would use neurotrophic factors, such as nerve growth factor in a method of inhibiting glial scars and promoting neuronal regeneration because NGF induces the differentiation and survival of neurons and is critical for the survival and maintenance of sympathetic and sensory neurons.

It would have been obvious to the person of ordinary skill in the art at the time of the invention was made to reduce GAG content in a glial scar and promote neuronal regeneration in a subject by inhibiting XT-I or XT-II using antisense oligonucleotides, ribozymes, DNA enzymes, or RNAi constructs.

The person of ordinary skill in the art would have been motivated to combine the teachings of Fawcett et al., Kleesiek, and Jen et al. in a method using DNA enzymes (and other inhibitors of mRNA) to XT-I or XT-II to inhibit glial scar formation and promote neural regeneration. Fawcett et al. suggest that inhibiting synthesis of GAG would promote neuronal regeneration, while Kleesiek et al. suggest inhibiting XT using knowledge of the XT cDNA sequence and Jen et al. suggest ribozyme, DNAzyme and antisense design for any DNA sequence.

The cited art suggests the methods of the instant claims. Fawcett suggests preventing synthesis of glycosaminoglycan (GAG) in a glial scar can promote neural regeneration (page 384, *How Might Axon Regeneration Be Promoted?* and *Preventing Synthesis of Inhibitory Molecules* sections). Kleesiek teaches XT is an enzyme in the biosynthesis of the glycosaminoglycan. A skilled artisan, knowing that preventing synthesis of glycosaminoglycan in a glial scar can promote neural regeneration, would seek methods to inhibit glycosaminoglycan. As skilled artisan might ask himself how he could inhibit GAG expression. A skilled artisan in seeking such information would come upon Kleesiek. Kleesiek teach the nucleic acid sequences of XT-I and XT-II and further suggest methods of gene therapy and methods of inhibiting XT-I and XT-II. A skilled artisan knowing that inhibiting XT-I and/or XT-II is capable of inhibiting the synthesis of GAG would seek methods of inhibiting XT-I and/or XT-II; agents used in gene therapy methods such as DNAzymes would be among those considered by a skilled artisan. Jen et al. is a review article about designing antisense oligonucleotides, ribozymes, and DNAzymes in which Jen et al. teaches “the DNAzyme can be made to

cleave virtually any RNA that contains a purine-pyrimidine junction" (page 312, col.2). A skilled artisan having the knowledge of Jen and Kleesiek and Fawcett would be able to make a DNAzyme to inhibit XT-I and/or XT-II, with the knowledge that these molecules would be agents that could inhibit GAG formation in glial scars. Accordingly, the examiner concludes that a skilled artisan would find that the cited art suggests inhibiting expression or activity of XT-I or XT-II using nucleic acid agents (RNAi, DNAzymes, Ribozymes, antisense) can and/or will reduce glycosaminoglycan content in a glial scar of a mammal.

There is a scientific rationale for using intrathecal, topical or local administration of agents to the glial scar. The Jen article discusses the issues required for RNAi, DNAzymes, Ribozymes, and antisense drugs to provide therapeutic effects, including drug delivery and localization. A skilled artisan, knowing the difficulties in delivering drugs to neurons and the requirement that the nucleic acid drugs listed above would require co-localization within the same intracellular compartments as the mRNA being targeted, would choose routes of administration such as intrathecal, topical or local administration. Therefore, the examiner concludes that a scientifically logical rationale exists for intrathecal, topical or local administration of RNAi, DNAzymes, Ribozymes, and antisense drugs.

Absent evidence to the contrary, an artisan would have expected success, because use of antisense oligonucleotides are well known in the art to inhibit expression of genes by inhibiting mRNA. From the teachings of Kleesiek , it seems possible to use the "knowledge of the cDNA of XT-I and XT-II to make gene therapeutic inhibitors of XT-

I and XT-II activity. Finally, Jen et al. suggest that any DNAzyme can be made, using knowledge of a given cDNA. Together, the prior art seems to provide all the known element required for using DNA enzymes for the inhibition of XT-I or XT-II.

Regarding the rationale for combining prior art elements according to known methods to yield predictable results, all of the claimed elements were known in the prior art and one skilled in the art could have combined the element as claimed by known methods with no change in their respective functions, and the combination would have yielded predictable results to one of ordinary skill in the art at the time of the invention. Each of the elements (cDNA sequence of XT-I and XT-II; theory of DNAzyme design; importance of XT in glial scar formation and neuroregeneration) are taught by Fawcett or Kleesiek or Jen. It would be therefore predictably obvious to use a combination of these three elements in a method using DNA enzymes (and other inhibitors of mRNA) to XT-I or XT-II to inhibit glial scar formation and promote neural regeneration. Furthermore, the specific DNA enzymes of SEQ ID NO:33 and 39 would be likewise obvious.

Therefore the method as taught by Fawcett et al. in view of Kleesiek and further in view of Jen et al. would have been *prima facie* obvious over the method of the instant application.

NEW GROUNDS OF REJECTION

Written Description (35 USC 112, first paragraph)

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claim 28 is rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

Instant claim 28 encompasses a method claim 26 which further comprises administering a genus of proteoglycan specific enzymes. Under the new Written Description Guidelines (March 25, 2008, Revision 1) the examiner is directed to determine whether one skilled in the art would recognize that the applicant was in possession of the claimed invention as a whole at the time of filing. The following considerations are critical to this determination:

a. Actual Reduction to Practice. In the instant case, the specification has not reduced to practice any proteoglycan specific enzymes.

b. Disclosure of structure. The specification does not provided any structure for the genus of proteoglycan specific enzymes.

c. Sufficient relevant identifying characteristics. The specification does not provided any identifying characteristics for the genus of proteoglycan specific enzymes.

d. The method of making the claimed invention is not well established, because the structure of the genus of proteoglycan specific enzymes is not described.

e-f. The level of skill in the art, and the predictability in the art are not well established and/or very predictable to a skilled artisan, with regard to generating the genus of proteoglycan specific enzymes, based on the state of the art and the instant application.

Therefore, the examiner concludes that there is insufficient written description of the instantly claimed method comprising a genus of proteoglycan specific enzymes.

Conclusion

No claims are allowed.

Examiner Contact Information

Any inquiry concerning this communication or earlier communications from the examiner should be directed to **Scott Long** whose telephone number is **571-272-9048**. The examiner can normally be reached on Monday - Friday, 9am - 5pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, **Joseph Woitach** can be reached on **571-272-0739**. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

/SDL/ Scott Long
Patent Examiner, Art Unit 1633

/Janet L. Epps-Smith/
Primary Examiner, Art Unit 1633